BMC Neuroscience

POSTER PRESENTATION Open Access

Modeling the effects of neuronal morphology on dendritic chloride diffusion and GABAergic inhibition

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From The Twenty Third Annual Computational Neuroscience Meeting: CNS*2014
Québec City, Canada. 26-31 July 2014

Gamma-aminobutyric acid receptors ($GABA_ARs$) are ligand-gated chloride (Cl^-) channels which mediate the majority of inhibitory neurotransmission in the CNS. Spatiotemporal changes of intracellular Cl^- concentration alter the concentration gradient for Cl^- across the neuronal membrane and thus affect the current flow through $GABA_ARs$ and the efficacy of GABAergic inhibition. However, the impact of complex neuronal morphology on Cl^- diffusion and the redistribution of intracellular Cl^- is not well understood. Recently, computational models for Cl^- diffusion and $GABA_AR$-mediated inhibition in realistic neuronal morphologies became available [1-3]. Here we have used computational models of morphologically complex dendrites to test the effects of spines on Cl^- diffusion. In all dendritic morphologies tested, spines slowed down longitudinal Cl^- diffusion along dendrites and decreased the amount and spatial spread of synaptically evoked Cl^- changes. Spine densities of 2-10 spines/μm decreased the longitudinal diffusion coefficient of Cl^- to 80-30% of its value in smooth dendrites, respectively. These results suggest that spines are able to limit short-term ionic plasticity [4] at dendritic GABAergic synapses.

Acknowledgements
Supported by the NSF/BMBF (US-German Collaboration in Computational Neuroscience, No. 01GQ1203A).

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Published: 21 July 2014

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doi:10.1186/1471-2202-15-S1-P138
Cite this article as: Mohapatra *et al.*: Modeling the effects of neuronal morphology on dendritic chloride diffusion and GABAergic inhibition. *BMC Neuroscience* 2014 **15**(Suppl 1):P138.